Interesting Electrocardiograms

1. ATRIAL PREMATURE CONTRACTIONS UNCOVER BIFASCICULAR DISEASE

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This 92 year-old woman, who was receiving digoxin and Isordil, showed the following ECG abnormalities, — on all sinus beats (the first, third and fourth beats in the first lead set of simultaneous leads I, II, III) there is complete right bundle branch block (RBBB) and a left axis deviation of -40°. With each of the frequent atrial premature contractions an increase in the degree of the left axis deviation occurs and the electrical axis becomes -58° (best seen in leads I, II, III, where the S wave deepens markedly). Thus left anterior fascicular block (LAFB) appears with an atrial contraction that is only mildly premature or early. The RBBB, of course, is also present. Bifascicular block (RBBB plus LAFB) replaces unifascicular block (RBBB alone) during the APC's.

The subtlety of this tracing lies in the measurement of the frontal plane electrical axis on the sinus beats and atrial premature contractions (APC's). On sinus beats simple left axis deviation (-40°) is present but it does not meet the criteria for left anterior fascicular block (which must reach -45° or more). The axis deviation of -58° in the APC's clearly meets this criterion. The usual aberrancy of the QRS associated with APC's is, of course, functional RBBB. The location of this functional IV conduction delay has recently been identified as occurring in the first 5 mm of the right bundle branch,—i.e., where the upper third of this bundle runs very close to the endocardium on the right side of the IV septum.

In this patient, RBBB of organic origin is already present on sinus beats. The left anterior fascicle of the left branch is seldom blocked by an APC unless it is also damaged by disease. A functional causation is rare. Thus when only a slightly premature APC produces LAFB and there is also a RBBB present during sinus beats, bifascicular damage of an organic type is likely. Possible future complete heart block must be considered and periodic Holter monitoring is indicated.
This 68 year-old man was admitted for differential diagnosis of either acute pancreatitis or acute myocardial infarction. The simultaneous leads II, aVR, and V1 are seen. Other leads were normal save for low T waves. Sinus P waves showing intermittent intra-atrial block (wide P waves, measuring 0.13 sec.) are present in all but the first three P waves on the strip where there is normal P wave duration. After the first three sinus beats, the heart rate slows considerably and if careful examination of the ST-T areas was not done and comparison made with the periods of sinus rhythm at the beginning and end of the strip, the presence of blocked atrial premature contractions (APC’s) deforming the ST-T waves (perhaps best identified in V1) would be missed. This would make for an erroneous diagnosis of SA node dysfunction with sudden onset of either sinus arrest or sinus exit block. The blocked APC’s are coupled to five sinus beats and penetrate and discharge the SA node each time, but only one sinus beat drops out and normal sinus rhythm then resumes for the last four beats. The SA node thus responds in healthy fashion to APC’s. Sudden rhythm changes relating to the function of the SA node are becoming more and more important. This tracing represents one of the commonest pitfalls in the electrocardiographic diagnosis of sudden bradycardias. For the first three sinus beats the rate is 74/min. and then, during the arrhythmia, the rate drops suddenly to 46/min. However, this is not due to sinus bradycardia. The SA node recovers each time (at a rate of 65/min.) after the APC invades the primary pacemaker and silences it for just one beat. The slightly slower rate of firing of the SA node after it has been silenced, or overdriven, is called a “warm-up” phenomenon,—i.e., after overdrive by the APC the pacemaker starts off firing more slowly than its normal firing rate (here 74/min.) but soon speeds up to its former, more rapid rate. The last four sinus beats on the tracing again occur at a rate of 74/min., testifying to a resumption of its normal, intrinsic rhythmicity. When the SA node is malfunctioning, the silencing effect, or overdrive phenomenon, is greatly exaggerated and would have caused a much slower recovery rate of the sinus rhythm after the APC’s ceased.
This 92-year-old woman, receiving digoxin and Isordil, entered the hospital with fever and a history of a recent myocardial infarction. As seen on simultaneous leads II, aVR and V1, there is a slow atrial (non-sinus) rhythm at 86/min. Note the negative P waves in lead II and positive P waves in aVR and V1 characteristic of this rhythm. The tenth beat is a ventricular premature contraction (VPC) with a retrograde P wave superimposed on its T wave. This is best seen in lead V1. The P vector of this retrograde excitation is the same as the P vector in the slow atrial rhythm in the first nine beats, attesting to a low atrial focus for this atrial rhythm. Both the low atrial pacemaker and the retrograde activation from the VPC depolarized the atria from the bottom of the chambers to the top.

Following the VPC there is a pause after which sinus rhythm returns at 83/min. The VPC depolarized the atria by retrograde excitation and thus wiped out, or silenced, the ectopic focus responsible for the slow atrial rhythm which fired at 86/min. This allowed the sinus rhythm to resume control of the cardiac rhythm at 83/min.

The appearance and disappearance of a slow atrial rhythm in this patient requires clinical evaluation. She had right bundle branch block (RBBB) and left axis deviation of -39° (full ECG not shown). She also was receiving a digitalis preparation raising the question of digitalis toxicity in a nonagenarian. The VPC’s are compatible with digitalis excess. The slow atrial rhythm, in contrast to paroxysmal atrial tachycardia, is not a sign of it however, being more suggestive of organic SA node disease. The IV conduction disease would, of course, reinforce the possibility of a dysfunctional SA node, since concomitant disease of specialized conduction tissues is frequent with pathology of the primary pacemaker. The basic sinus rate of 83/min. however does not suggest the sick sinus syndrome in which sinus bradycardia is more likely. However, the patient was febrile and this sinus rate might be higher than would be seen without fever. Close follow-up of her rhythm fluctuations is therefore advisable.